

The operations for the relief of ascites all have for their object the establishment of a collateral circulation between the portal and general venous systems. This is produced through the establishment of peritoneal adhesions. To effect this result the omentum is generally utilized in one or other of the following ways: (1) It may be sutured to the parietal peritoneum alone. (2) It may be fixed under the skin of the abdominal wall. (3) It may be sutured between the liver and diaphragm. The parietal peritoneum may be separated from the muscles around the abdominal incision and the omentum grafted into the prepared space. At the time of the operation it is customary to scrub the surface of the liver, diaphragm, and spleen to promote their subsequent adhesion.

Montgomery reports two cases on which he himself has operated. In one case the ascites apparently was cured, as it had entirely disappeared six months after operation. The other case was only slightly relieved.

Since the analysis by Packard and Le Conte of 22 cases collected from the literature, Montgomery has found records of 10 others, including his own, making a total of 38 cases. The result in these cases was as follows: 10 deaths, or 26.3 per cent.; 9 unimproved, or 23.6 per cent.; 4 improved, or 10.5 per cent.; 15 recovered, or 39.4 per cent.

On Adrenalin Glycosuria and Other Forms of Glycosuria Due to the Action of Reducing Substances and Other Poisons on the Cells of the Pancreas.—Some months ago the experiments of F. BLUM on adrenal diabetes were noted in this department. Blum found that the subcutaneous injection of an aqueous extract of adrenal glands produced glycosuria in twenty-two out of twenty-five animals experimented upon. Recently Herter (*American Medicine*, May 10, 1902, p. 771) has carried out some instructive experiments showing the effect that adrenalin chloride and solutions of other reducing substances have when painted over the surface of the pancreas.

Commercial aqueous solution of adrenalin chloride 1 : 1000 was used in the research. Herter first showed that the subcutaneous, intravenous, and intraperitoneal injections of adrenalin chloride into dogs were almost invariably followed by glycosuria. Peritoneal injection, other conditions being equal, produced the most marked glycosuria, 10 per cent. or more of sugar not being uncommon. The glycosuria lasts usually somewhat less than twenty-four hours. It was important to find whether the more marked results following intraperitoneal injection of adrenalin chloride were due to the opportunity for more direct action of this substance on the pancreatic gland. Accordingly the pancreas was exposed in a number of normal dogs and adrenalin applied directly to the presenting surface by means of a brush. It was demonstrated that marked glycosuria follows the application of small quantities of adrenalin to the pancreas, quantities which, when applied locally to other parts of the body, either give rise to no excretion of sugar or to a trivial glycosuria.

The next problem was to determine how the local application of adrenalin to the pancreas produced glycosuria. It was first shown by experiment that it was not due to vascular disturbance. Adrenalin chloride is known to be a very powerful reducing substance, and it was decided to ascertain the effect that other reducing substances had when applied directly to the pan-

creas. The thought suggested itself that the use of such reducing substances might interfere with oxidation within the pancreatic cells. Local applications of potassium cyanide were first tried and constantly produced a glycosuria. Similar results were obtained with sulphurous acid, ammonium sulphide, sulphuretted hydrogen, illuminating gas, carbon monoxide and several other reducing substances. A number of similar experiments were carried out with substances which do not reduce, such as sodium chloride, sodium hydroxide, ferric chloride, hydrochloric acid, bromine water, etc., without the production of glycosuria in any instance.

Herter concludes from these experiments that there is a close relationship between the reducing power of a substance and the capacity of this substance to induce glycosuria when applied directly to the pancreas. He believes that the amount of oxygen which these substances remove from the cells of the pancreas is sufficient to cause a disturbance of the function of the gland.

Histological examination of the gland tissue at the height of the glycosuria fails to show any recognizable lesion. Herter does not believe that the glycosuria is due to disturbance of the function of the islands of Langerhans.

It is pointed out that these experiments are of clinical importance, in that they probably throw considerable light on several varieties of human glycosuria and diabetic cases. Herter suggests that the glycosurias following conditions of asphyxia, as after epileptiform seizures, may be due to interference with oxidations in the pancreas. So also with the glycosuria following carbon monoxide poisoning.

Contribution to the Study of the Myospasms: Myokymia, Myoclonus Multiplex, Myotonia Acquisita, Intention Spasm.—WALTON (*Journal of Nervous and Mental Disease*, July, 1902, p. 402) draws attention to the fact that there is much confusion in certain text-books, and often on the part of writers, in the classification of the various anomalies of the neuromuscular mechanism which gives rise to involuntary spasmodic movements. The writer endeavors by means of the reports of illustrative cases to clear up certain misconceptions which have led to error in the classification of these interesting cases. His conclusions are as follows:

(1) The term *myoclonia*, as a collective designation for the unrelated disorders, should be discontinued.

(2) The term *myokymia* should be limited to cases showing, without hereditary or congenital history, widespread muscular quivering, without atrophy or other indication of progressive degeneration of the nervous system, without constitutional symptoms, and without sign of present infectious or other acute disease. Cases should not be excluded, however, on account of preceding or introductory symptoms pointing to disease of the lower neurons, if such disease has either disappeared or come to a standstill.

(3) The term *myoclonus fibrillaris multiplex* should not be applied to such cases, since it suggests a relationship between myokymia and the paramyoclonus multiplex of Friedreich, with which it has nothing in common.

(4) *Myoclonus multiplex* (the prefix *para* seems superfluous) should be used to designate bilateral clonic spasms involving whole muscles or groups of muscles, generally those attached partly or entirely to the trunk.